the wetland, and leaving the wetland. Water was collected at various times of the year and experiments were repeated. Surprisingly, water entering the wetland and midway through had no effect on early development but water leaving the wetland was largely lethal. This suggests that unidentified environmental contaminants are leaching out of the reclaimed wetland and entering surface water supplies. Far more lethality and developmental abnormalities were observed in the dejellied embryos versus those with their jelly coat intact, which suggests that the protein matrix of the jelly coat provides some protection from environmental contaminants. Subsequently, we found that a known estrogenic environmental contaminant (nonylphenol) showed similar effects on dejellied versus jelly coated embryos. We hope this work highlights the need to not only reclaim wetlands from existing farmland, but also to monitor these sites after they have been established.

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Edn1/Ednra pathway in *Xenopus* neural crest development

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The neural crest (NC) develops at the border between neural plate and the prospective epidermis in vertebrate embryos. NC cells are highly migratory and generate a number of derivatives including neurons, pigment cells, craniofacial cartilage, endocrine cells, etc. Numerous studies have demonstrated that many signals (BMP4, Wnt, FGF, etc.) are involved in the induction of this tissue. However, the participation in neural crest specification of other cell signaling pathways has not been established yet. In this work, we have analyzed the expression and participation of the Endothelin1/Endothelin receptor A cell signaling pathway during *Xenopus laevis* development. We report the cloning and expression pattern of Ednra cDNA. Ednra is expressed at the neural plate border from early neurula stage, in the NC cells during migration, and in branchial arches and the otic vesicle. We analyzed the role of Edn1/Ednra pathway in NC development by conditional gain- and loss-of-function approaches using mRNA microinjection, morpholino-oligonucleotides, and the specific inhibitor of Ednra BQ123. We also present embryological evidence showing Edn1/Ednra pathway is also involved in the maintenance of NC specification and cell survival. Our results show that Edn1/Ednra cell signaling pathway is required for the induction and migration of neural crest cells in *Xenopus* embryos. Funding: CIUNT, PICT10623, ICM P02-050, PICTOUNT367, PIP6278, and UNSTA.

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Banded hedgehog and Gli intracellular factors control *Xenopus* neural crest specification

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Secreted morphogens of the Hedgehog (Hh) family in *Xenopus* are Sonic hedgehog, Cephalic hedgehog, and Banded hedgehog. They regulate a wide range of developmental processes such as nervous system and limb patterning. In this work, we revised the expression pattern of Banded hedgehog (Bhh) and components of the intracellular signaling cascade during early development of *Xenopus laevis* embryos. The double in situ hybridization analysis showed that Bhh is expressed during neurulation at the lateral border of neural plate in territories that overlap with the expression of Gli transcription factors and neural crest markers. In order to evaluate the participation of Bhh pathway in neural crest development, we carried out gain- and loss-of-function approaches by directed microinjection of Bhh mRNA, its dominant-negative and morpholino oligonucleotides. Results showed that the overexpression of Bhh leads to an increased expression of neural crest markers and Gli transcription factors. On the other hand, the dominant-negative construct of Bhh and the morpholino oligonucleotide reduced the expression of neural crest markers, indicating Bhh signaling is required for neural crest specification. Additionally, the overexpression of Gli3 produced an expansion in neural crest territory mimicking the effect produced by Bhh gain of function. Our results show that Bhh signaling and Gli transcription factors are participating in the early neural crest development. Funding: CIUNT, PICT10623, ICM P02-050, PICTOUNT367, PIP6278, and UNSTA.

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A mutant with defective temporal coordination of uterine and vulval development in *C. elegans* is associated with reciprocal signaling defects

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Many biological functions require interactions between different organs whose development must be coordinated both spatially and temporally. For example, egg laying in *C. elegans* requires that a connection form between the lumens of the uterus in the gonad and the vulva in the extra-gonadal epithelium during organogenesis. A *cog-3(ku212)* mutant appears to form no connection between the vulval and the uterine lumens because the uterine lumen develops with a temporal delay relative to the vulva and thus is not present when the connection normally forms. The lack of temporal synchronization between the vulva and the uterus is not due to precocious or accelerated vulval development. Instead, gonadogenesis is delayed relative to